Leprosy

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WITH the advances made in our present knowledge of the interdependence of the functions of organs in the production of normal health, the cause and effect in disease is a problem that must ever be changing as our knowledge increases. When I was a student it was taught that fibroids were the *cause* of sterility. We now believe that sterility is the *cause* of fibroids. Bad teeth are not so much the *cause* of ill-health, as that ill-health is the *cause* of bad teeth and it took a great war to change our views on this question. With this problem of *cause* and *effect* in disease I perhaps need not apologise for wishing to talk to you for a few minutes on what is one of the oldest diseases in the world, viz., leprosy. It is a disease from which the people of this province suffer as much as any other province in India. Notwithstanding the stimulus given to the treatment of this disease in the last few years, I would take you with me in a review of our present knowledge of this disease and consider whether we should not do a little stock-taking of our knowledge and view it from another angle.

What has impressed me with regard to this disease are certain accepted dogmatisms, which are difficult to reconcile with our present-day medical knowledge.

The first point of difficulty is that ever since Hansen discovered in 1874 the acid-fast red staining rods in the tissues of cases of leprosy which were, and are still, referred to as Hansen's bacilli, the medical profession has endeavoured to fit in the different clinical manifestations of this disease with the theory that they are the result of an infection by this so-called bacillus. But the bacteriologists have not yet proved that it is a bacillus. It can neither be isolated nor cultured, it cannot be injected into another animal and produce the disease and Koch's postulates as to the proof of any bacillus being the cause of a disease have not been fulfilled in this respect. *Secondly.*—In trying to fix the incubation period of this disease, we are told that periods of four or five years must be considered as a normal period, and that this apparently prolonged incubation

period is due to the insidious manner in which the disease develops. This prolonged incubation period is not a feature common to any other disease known to us which is supposed to be caused by infection. Even hydrophobia or rabies, which has a very long incubation period as compared to other diseases, does not approach this term which we are asked to believe is characteristic of this disease. *Thirdly.*— The contagiousness of leprosy. If you read the examples given of the contagiousness of this disease you are struck with one factor, and that is the want of scientific proof of this contagiousness.

Such general statements as that people who have worn the clothes of sufferers from leprosy have subsequently developed the disease, or that persons who have lived in rooms occupied by such people have been infected, leave one with the impression that the medical profession finds it somewhat difficult to produce convincing evidence on this point. In this country where leprosy is common, one may well retort with the enquiry how many people who have worn the clothes of those suffering from or have lived in the same room with them have *not* developed leprosy?

Moreover, we know that conjugal relationships do not produce the figures of infectivity which we should expect from such close and intimate contact. The medical experts labour to place this figure at 2 to 5 per cent. which, when compared to the frequency with which blood relations are infected (up to 50 per cent. in one census), is a piece of negative evidence which should make us hesitate to be confident that our established views are quite consistent with the facts that are presented to us.

Omitting minor manifestations of this disease, which it is difficult for modern medical knowledge to reconcile with the established views, we have sufficient and efficient reasons for doubting the correctness of the established views on leprosy, namely:—

(1) that we have failed to prove that Hansen's bacillus is the causative agent of the disease;

(2) that we have no positive proof that the disease is contagious; and

(3) the long incubation period required for its development.

These serious difficulties justify us in trying to find some other angle from which to view the disease and one which might be more consistent with the facts as we find them.

The history of the disease takes us back to many hundreds of years B.C., and formerly it was as common in

Europe as in the tropical countries. But in England it began to decline in the latter part of the 14th century and nearly disappeared by the end of the 16th century. It also declined in the other European countries, but even to this day remains in certain parts of Norway, the Baltic Provinces, parts of Russia and Turkey.

What is supposed to have been the cause of this decline? We read that at the end of the 15th and beginning of the 16th century, the social conditions of the people in Europe improved. Their dietary, which was perhaps as primitive as that which is extant in Africa or parts of India to-day, shewed a marked advance with the better social conditions of the people. But the fact that the disease still occurs in parts of Europe, such as Norway, Sweden, parts of Russia and the Balkan Provinces, leads us to infer that the social conditions under which the people live in these countries are still a factor in keeping up the incidence of the disease. And if one investigates the social conditions one finds a common factor that the country is still poor in these localities and that, being poor, the dietary of the people themselves is of a limited nature.

If we take into consideration the countries shewing the greatest prevalence of the disease at present we find this one common factor—the primitive conditions under which the people live with the concomitant limited nature of their dietary—and that Central America, Equatorial Africa, India and China, are the countries which shew the greatest incidence of this disease. If we investigate the conditions in such a country as Norway, where it still exists in Europe, we find it not in the large towns, but in the poor population living on the sea-coast and where the dietary consists largely of fish. In Japan the disease is becoming less, and it may be that with the improvement of the social conditions in Japan and the change in the dietetics of the Japanese the disease is slowly being eradicated.

In this country and in this province of Bihar and Orissa can we say that the social conditions under which the people live are a factor in the incidence of this disease? The customs and prejudices of the people combined with the limited dietary at their disposal would lead us to infer that if the necessary predisposing idiosyncrasy to this disease is forthcoming, the means for its propagation is present. From which you will all obtain an idea of the angle from which I am trying to view this disease, *viz.*, that it is a deficiency disease.

To be brief, I will summarise the points for consideration

for viewing this disease as a deficiency disease, if I may so call it.

(1) The clinical manifestations are such that an affection of the nerves and the periphery nerves are the predominant feature. It is a disease of the nervous system with generally a predisposition to affect the nerves as distinguished from the spinal cord or brain. If Hansen's bacillus or any other bacillus is the root cause of the disease why should not the affection be generalised? why should not the viscera of the body be affected? And yet clinically we find that, excepting the skin manifestations and the trunks of nerves, the manifestations in other parts of the body which may be affected can be attributed to a pathological nervous entity.

(2) How then can we reconcile this as being an affection of the nervous system with the skin lesions that are the predominating feature in so many cases? We have plenty of evidence in clinical cases that the nerves can become affected without any skin lesions, but we have very little or no evidence that where the skin lesions are predominant the nerves fibres are not affected in this area. If we take the superficial erythematous lesion, do not we find that this lesion is generally anæsthetic, and in this skin lesion what if the nerve fibrils that are affected are connected with trophic functions? Will this not account for this picture, *viz.*, that the nerve fibrils affected are concerned either directly with trophic functions, or if not directly then indirectly by nerve fibrils supplying the finer blood vessels and so cause the skin lesions through disturbances of the circulation?

If, then, we say that the skin lesions are of a trophic variety either caused directly through nerve disturbances or indirectly through changes in the capillaries-rarefaction and absorption of bones is easily explained by interference with the nourishment of the bone and its periosteum.

(1) Other evidences of affection of the nerves in this disease are that cases occur similar to Herpes Zoster.

(2) Tingling and numbness may be a premonitary symptom before any apparent changes of the tissues.

(3) In some cases blisters on the hands and feet may be the first manifestations of the disease.

(4) The perforating ulcer is a common clinical manifestation in this disease.

(5) The ulcers that one sees in this disease may certainly be classified as trophic ulcers.

How shall we explain the nodular variety of leprosy? If we admit that the superficial skin lesions are the result of trophic disturbances we find that according to the

resistance of the tissues and the vascularity of the part so is determined a radially spreading superficial lesion or a circumscribed nodule. Hence the predilection of the nodular manifestation for the skin of the face and ears and the absence of nodules in the palms of the hands or soles of the feet.

Then how to explain the acid-fast rods that are known as Hansen's bacilli? There are gross changes going on in the tissues, and in such changes there is probably chemical transformations incidental to pathological causes, and this being so, can we not infer that these acid-fast rods are due to a metamorphosis of the tissues ; changes of staining capacity in the tissues which are the *result* of the disease and not the *cause* of it?

When discussing this disease with a doctor, I was informed that the belief of the *kavirajs* was that these red staining rods are the result of the disease and not the cause ; so that on this point *kavirajs* and I have something in common ; but whether I am in bad company or they are in good company is a matter for others to decide. When we consider, however, that hydrophobia or rabies are responsible for the Negri bodies in the brain and that these bodies can be produced by excitants other than rabies poison, we must recognise that just as the Negri bodies are the *result* and not the *cause* of the disease, so it may be that the so-called Hansen's bacillus is not the *cause* of this disease but the *result* of it. Moreover, the vagaries of the presence or absence of these so-called Hansen's bacilli in leprosy cases would lead one to infer that they are rather products of chemical alterations of the tissues, than that they bear any relation to its clinical manifestations. With these somewhat cursory criticisms I put forward the view that leprosy should be classified as a deficiency disease. That it is an imbalance of the metabolism of the tissues due to some defect in diet which, by the absence or diminution of important vitamines or food accessories cause the formation of toxic products and that these toxic products affect the peripheral nerves. It is now held that vatamines B and B1 are important factors in maintaining the health of the nerves, and it has recently been shown by Mellanby that vitamine A is also required in an abundant supply for the same purpose. The degeneration of the nerve fibres may be selective for the class of fibrils affected ; and if we maintain that there are definite trophic nerve fibres, this would be consistent with the lesions found in leprosy. And even without straining our imagination that the skin

lesions are the results of disease of trophic nerve fibrils, we have the picture of the skin manifestations in pellagra, which is now classified as a deficiency disease.

Compare these two diseases, pellagra and leprosy, and we find some common factors to both diseases.

(1) The exacerbations and periods of quiescence of the disease.

(2) The skin affections.

(3) The nervous affections : in pellagra chiefly cord and brain ; in leprosy peripheral nerves.

(4) An acute form occurs in both diseases : in pellagra known as pellagra typhus; in leprosy known as leprous fever.

I have stated above that the imbalance of the metabolism of the tissues may be due to a diminution or absence of important vitamines ; but as we know so little about these food accessories, it may well be that an excess of one vitamine may result in causing pathological entities just as much as the diminution or absence of them—or to put it in other words, an interdependence of the different vitamines may be necessary for normal health.

How does our knowledge of the distribution of this disease fit in with this theory of a deficiency disease ?

Take the disease as it occurred in England before the 16th century, when the food of the people was poor-salted meat, fish, or black rye bread, etc. With the end of the dark ages the social conditions of the people improved and leprosy decreased. This I grant you may have been due to other improvements than dietary, but it may help in reviewing the disease as a whole. Where does the disease exist now, but in those parts where social conditions are poor and the dietary is poor, and restricted or dried foods are predominant in the dietary ?

To come down to this province of Bihar and Orissa, the diet of the masses in those localities where leprosy is most abundant is almost of the same nature for every meal and for every day. The variety of food-stuffs obtained by these masses is very limited. The cereals, pulses and even vegetable foods are preserved dry, and with the absence of fresh food, such as milk, meat, fruits and vegetables, which these people are unable to obtain, one may well appreciate the fact that food kept in preservation may lose those anti-neuritic qualities which are necessary for the preservation of normal, healthy nerves. It would appear that in this province certain classes of aboriginals with no caste principles and who will eat anything, birds, snakes, etc., do not

suffer with this disease as compared with those aboriginals who have cultivated sufficient land to provide enough food for the whole year and who have taken to dried or preserved cereals as their main food supply like the Hindus or Muhammadans.

We may also put in the same category the people of the countries who live on dried fish as a staple food ; the fact that it is dried or preserved may affect its food value in the same way as preserved rice or cereals in this country. We may now consider why in the same village some people escape leprosy whereas others contract it, and why the children of leprous parents are more susceptible to the disease. If there is a disturbance of the metabolism of the tissues owing to want of necessary food accessories, which result in toxic substances being formed in the tissues, there must also be a personal idiosyncrasy of the subject to affections of the nerves—there must be, as in all nervous diseases, an inherent susceptibility which susceptibility is familial.

All persons suffering from syphilis do not develop locomotion ataxia, and all people who eat the vetch lathyrus sativus do not contract lathyrism ; similarly, all people who live on a defective diet do not contract leprosy. This inherent susceptibility to nervous disease will explain the figures given above that conjugal relationships do not produce the figures of infectivity, but that blood relationships do.

A factor in the causation of affection of the nerves may be stated to be the absorption of minute quantities of poison. If we take the polyneuritis that occur in alcoholics we find that it is the constant imbibing of the weaker alcoholic drinks that cause the disease, and in the ascending neuritis of lead poisoning it is the absorption of minute quantities of the poison that cause the trouble. Compare this with the slow formation of a poison as a result of an imbalance of metabolic processes in the body and we have an explanation of the prolonged incubation period which has been attributed to this disease. It would appear to me that many of the phenomena of leprosy can be explained by this theory with less straining of our imagination than any infectivity theory.

Take for instance the case of Father Damien, who contracted leprosy after working for years in one of the Pacific Islands where leprosy was rife. He is supposed to have at last contracted the disease, but what did Father Damien have to eat on this island ? He had to eat probably what the natives of the island ate, and he at last manufactured sufficient poison owing to a defective dietary to affect him.

Moreover, I believe I am correct in saying that there were also nursing sisters with him on that island—but not one of them contracted the disease. So that the susceptibility of the individual or the threshold of resistance to such poisons varies in different individuals.

And similarly with the children of cases of leprosy, who doubtless have a lower threshold of resistance, which disability is inherited from their parents ; these children, if removed from their surroundings, and given special dietary, may not contract the disease, and one of the most useful works done by the Missions in India is the rescue of these children from their surroundings. In this province the Mission Leper Colony at Purulia is an object lesson of such a good work.

Furthermore, we must remember that youth is the most suitable age to contract this disease and this is perhaps more explicable than the theory that with a growing body the need for accessory food-stuffs is at its highest. The result of the intensive treatment now being administered for leprosy would appear to favour the theory that it is a deficiency disease. Cases treated as out-patients have not produced the same results as those treated as in-patients, where, in addition to medicines, the general health of the patient is improved and, incidentally, a change in dietary is given. I remember in this very town of Darbhanga, which has an out-patient leprosy clinic, asking the doctor in charge how many cases within the last three years had become " symptom free." The reply was " five," of which " two " had relapsed and the other " three " untraced. This might be cheerful news, but it certainly was not intoxicating.

On the other hand, take the figures which were given me by the Mission Leprosy Colony at Purulia, for 1930. Of the treatable cases it is recorded of those attending as out-patients two became symptom free ; but of the in-patients there were as many as 41 cases that became symptom free. The advance in knowledge on vitamines or food accessories is remarkable at the present time, and although we are prone to apply new discoveries to old diseases, yet on the principle of trying to find one common factor to explain all the manifestations of the disease, this factor of a deficient dietary seems to me to warrant further investigation in this disease of leprosy.